

BACTERIAL ISOLATES AND THEIR ANTIMICROBIAL RESISTANCE PATTERNS IN NEONATAL SEPSIS RECORDED AT HEVI TEACHING HOSPITAL IN DUHOK CITY / KURDISTAN REGION OF IRAQ

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Submitted 24 February 2019; accepted 24 July 2019

ABSTRACT

Background: Neonatal sepsis is one of the commonest causes of neonatal mortality in the developing world. There is a continuous change in the patterns of microbial flora and their antimicrobial susceptibility. The aim of this study was to determine the microbial agents causing neonatal sepsis, the susceptibility of these microorganisms to the commonly used antimicrobial agents at Hevi Hospital and to compare the causative agents during the last four years.

Methods: A retrospective study was conducted over a period of four years. From the recorded data of archives of Hevi Teaching Hospital from 2014 till 2017, a total of 1058 blood cultures were taken. The positive and negative cultures were 536 and 522 respectively. From the positive cultures, 555 pathogens were isolated.

Results: The present study revealed that the female to male ratio for neonatal sepsis was 1.5:1 and the most common microorganisms isolated were Coagulase-Negative Staphylococci consisting (58.5%) of cases; followed by *Staphylococcus aureus* (16.6%) and *Escherichia coli* (7.6%). The higher percentage of neonatal sepsis (30%) was recorded in 2017. There is no statistical significant difference regarding the causative microorganism for the early and late neonatal sepsis except for the *Enterococcus* species; with their highest percentage among late neonatal sepsis.

Conclusions: This study revealed that resistance against many commonly used antibiotics have been increased which limits the options for treating of neonatal sepsis. This is resulted from uncontrolled use of the antibiotics and lack of policies and guidelines for their use in public health.

Duhok Med J 2019; 13 (2): 84-95.

Keywords: Antibiotic, Resistance, Neonatal sepsis

Neonatal sepsis is a serious infection that occurs in infants in their first 28 days of life and is a leading cause of morbidity and mortality of newborns. It is considered be a cause of 26% of all neonatal deaths worldwide^{1,2}. The Incidence of neonatal sepsis varies from one country to other but is more common in developing countries,

where it is responsible for about 30-50% of the total of neonatal deaths^{3,4}. It comprises a several systemic infections of the newborns including septicemia, osteomyelitis, meningitis, pneumonia and arthritis^{5,6}.

Depending on the time of starting of the infection, neonatal sepsis has been categorized into early-onset sepsis (EOS) and late-onset sepsis (LOS). Sepsis that

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occurs in the first three days of life called EOS and is often transmitted by vertical transmission from mothers to infants during the intra-partum period⁷. While LOS defined as infection occurring after 72 hours of birth; due to transmission of infection from neonatal intensive care. EOS in term newborns are mainly caused by Group B Streptococci; however Gram-negative bacteria are not rare. A combination of benzyl penicillin and intravenous gentamicin are most commonly used as a guideline in 125 UK hospitals⁸. Most cases of LOS are attributable to Group B Streptococci and *Staphylococcus species*, while gram negative microorganisms (*Klebsiella spp* and *Escherichia coli*) are responsible for about one third of cases⁹.

Early detection and correct management of neonatal sepsis by good care and rational antimicrobial can decrease mortality rate from this disease¹⁰.

Antimicrobial resistance recently considered as a major problem worldwide, about half of the microorganisms that cause severe neonatal bacterial infections reported to be resistant to the first-line (ampicillin or penicillin, and gentamicin) and to second-line (third-generation cephalosporins) treatments that the WHO has been recommended¹¹. The first estimation of neonatal deaths due to the neonatal sepsis that results from the antimicrobial resistance was published in 2016¹².

This study was conducted to detect the bacterial agents causing neonatal sepsis (early and late), to determine the antimicrobial susceptibility patterns of the bacterial isolates and to assess the available choices for empirical antimicrobial therapy in patients with neonatal sepsis.

PATIENTS AND METHODS

The antibacterial susceptibility and microbiological data of this study has been obtained from the records of the Microbiology laboratory of Hevi Pediatric Teaching Hospital. A retrospective survey study was carried out by taking the records of patients that revealed positive bacterial blood cultures and their antibacterial susceptibility results from archives for a period of four years (2014, 2015, 2016 and 2017). A total of 1058 blood cultures were recorded. The positive cultures and negative cultures were 536 and 522 respectively. From the positive cultures 555 pathogens were isolated. Neonates age less than 8 days were considered as EOS and those aged 8-90 days were LOS¹³.

The cultures were conducted by collecting blood (1-3ml) in blood culture bottles specific to BacT Alert instrument or in specific bottles manufactured for manual incubation and were incubated for 5 days. Few drops from culture positive bottles were collected by sterile needles and further streaked on media agar plates (blood, chocolate and MacConkey) and incubated for 24h. The isolated bacteria were identified based on their colony morphology, culture characteristics and biochemical reactions. Antimicrobial susceptibility testing was performed on Muller-Hinton agar (except for streptococci which were performed on blood agar plate) using disk diffusion method (Kerby-Bauer disc diffusion) against different antimicrobial agents according to CLSI standards¹⁴. The antibiotic discs consisted of Penicillin, Ampicillin, Amoxicillin/ clavulanic acid, Piperacilin, Cephalothin, Cefixim, Cefotaxime, Ceftazidime, Amikacin, Gentamicin, Ciprofloxacin, Norfloxacin, Imipenem, Vancomycin, Erythromycin,

Azithromycin, clindamycin and Trimethiprim.

STATISTICAL ANALYSES

The data were coded, computerized and analyzed by SPSS for Windows, version 22, identified by frequencies and percentages. Statistical significance was calculated using P value. P value less than 0.05 was considered as a significant result.

RESULTS

Five hundred and thirty six cases (50.6%) out of 1058 their blood cultures yielded the growth of bacterial pathogen so confirmed as having neonatal sepsis. The total number of isolated bacterial microorganisms was 555; 125 (22.5%) from 2014, 134 (24.1%) from 2015, 128 (23.1%) from 2016 and 168 (30 %) from 2017 (Figure 1). Male constitute 329 (59

%) and female 226 (41 %). Male to female ratio for neonatal sepsis was 1.5:1.

The most common bacterial pathogen caused the neonatal sepsis during all of the four years was Coagulase-Negative Staphylococci consisting (58.5%) of cases; followed by Staphylococcus aureus (16.6%) and Escherichia coli (7.6%) (Figure 2).

EOS constitute (30.8%) of cases. Coagulase-Negative Staphylococci was the commonest pathogen affecting both EOS and LOS with no significant difference (p value > 0.05).

Escherichia coli was the highest among LOS, but without any significant difference (p value > 0.05). Enterococcus species was more in LOS with a significant difference (p value < 0.05), (Table 1).

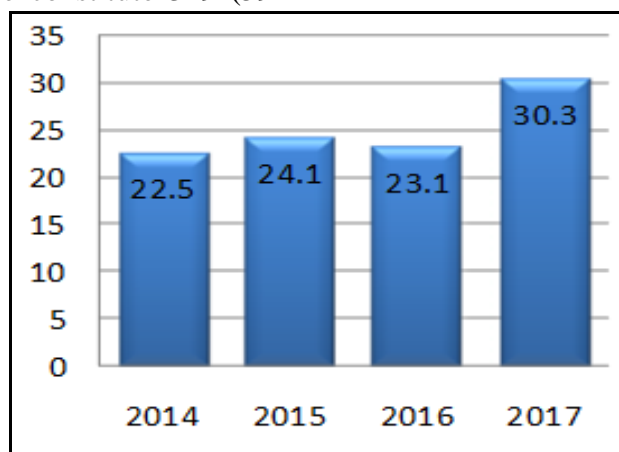


Figure 1: Percentage of Neonatal Sepsis During the Years 2014, 2015, 2016, and 2017

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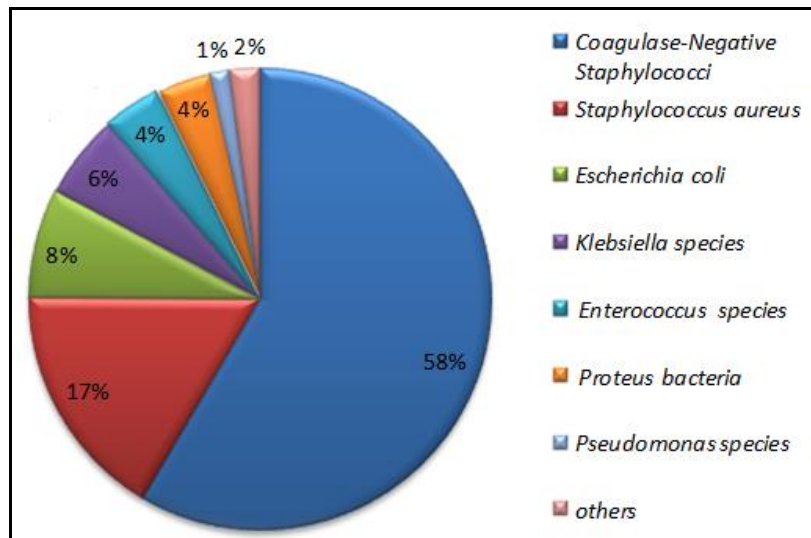


Figure 2: Percentages of the Isolated Microbial Pathogens

Table 1: Distribution of Pathogenic Microbial Agents in Early and Late Onset Sepsis

Microorganism		Early NS (1-7days) n(%)	Late NS (8-90 days) n (%)	Total n(%)	p- value
Gram positive bacteria	<i>Staphylococcus aureus</i>	27(15.9)	65(16.9)	92(16.6)	>0.05
	Coagulase-Negative Staphylococci	97(57.1)	228(59.2)	325(58.5)	>0.05
	<i>Enterococcus</i> species	2(1.2)	21(5.5)	23(4.1)	<0.05
	<i>Bacillus</i> species	0 (0)	4(1)	4(0.7)	>0.05
	Diphtheria species	0(0)	2(0.5)	2(0.4)	>0.05
	<i>Streptococcus agalactiae</i>	1(0.6)	0(0)	1(0.2)	>0.05
	Viridians streptococci	1(0.6)	0(0)	1(0.2)	>0.05
Gram negative bacteria	<i>Escherichia coli</i>	16(9.4)	26(6.8)	42(7.6)	>0.05
	<i>Klebsiella</i> species	15(8.8)	18(4.7)	33(5.9)	>0.05
	<i>Proteus</i> species	6(3.5)	15(3.9)	21(3.8)	>0.05
	<i>Pseudomonas</i> species	5(2.9)	3(0.8)	8(1.4)	>0.05
	<i>Brucella</i> species	0(0)	2(0.5)	2(0.4)	>0.05
Fungi	<i>Candida</i> species	0(0)	1(0.2)	1(0.2)	>0.05
Total		170(100)	385(100)	555(100)	

Coagulase-Negative Staphylococci, *Staphylococcus aureus* and *Escherichia coli* are more common among male than female (34.9% and 23.6%), (9.2% and 7.4%) and (4.7 and 2.9) respectively; but without statistically significant difference

(p value>0.05). On the other hand *Klebsiella species* are the highest among female (2.7% and 3.2%), this result was not significant statistically at p value >0.05 (Table2).

Table2: Distribution of Pathogenic Microbial Agents in Neonatal Sepsis According to Sex

Microorganism		Male n(%)	Female n(%)	Total n(%)	P value
Gram	Coagulase-Negative Staphylococci	194(34.9)	131(23.6)	325(58.5)	>0.05

positive bacteria	<i>Staphylococcus aureus</i>	51(9.2)	41(7.4)	92(16.6)	>0.05
	Viridians streptococci	1(0.2)	0(0)	1(0.2)	>0.05
	<i>Enterococcus</i> species	13(2.3)	10(1.8)	23(4.1)	>0.05
	diphtheria species	1(0.2)	1(0.2)	2(0.4)	>0.05
	<i>Streptococcus agalactiae</i>	1(0.2)	0(0)	1(0.2)	>0.05
	<i>Bacillus</i> species	3(0.5)	1(0.2)	4(0.7)	>0.05
	<i>Proteus</i> species	14(2.5)	7(1.3)	21(3.8)	>0.05
Gram negative bacteria	<i>Escherichia coli</i>	26(4.7)	16(2.9)	42(7.6)	>0.05
	<i>Pseudomonas</i> species	7(1.2)	1(0.2)	8(1.4)	>0.05
	<i>Brucella</i> species	2(0.4)	0(0)	2(0.4)	>0.05
	<i>Klebsiella</i> species	15(2.7)	18(3.2)	33(5.9)	>0.05
Fungi	<i>Candida</i> species	1(0.2)	0(0)	1(0.2)	>0.05
Total		329(59.2)	226(40.8)	555(100)	

Coagulase-Negative Staphylococci is the leading pathogen in all four years with a highest level in 2014 and 2016 (74.4% and 56.8% respectively). *Escherichia coli* (15.4%) were more during 2015.

Staphylococcus aureus (20.7%) and *Enterococcus species* (8.5%) were with

their highest percentage in 2016. On other hand, *Proteus bacteria* (7.1%) and *Klebsiella species* (8.8 %) were with a higher percentage in 2017 (Figure 3).

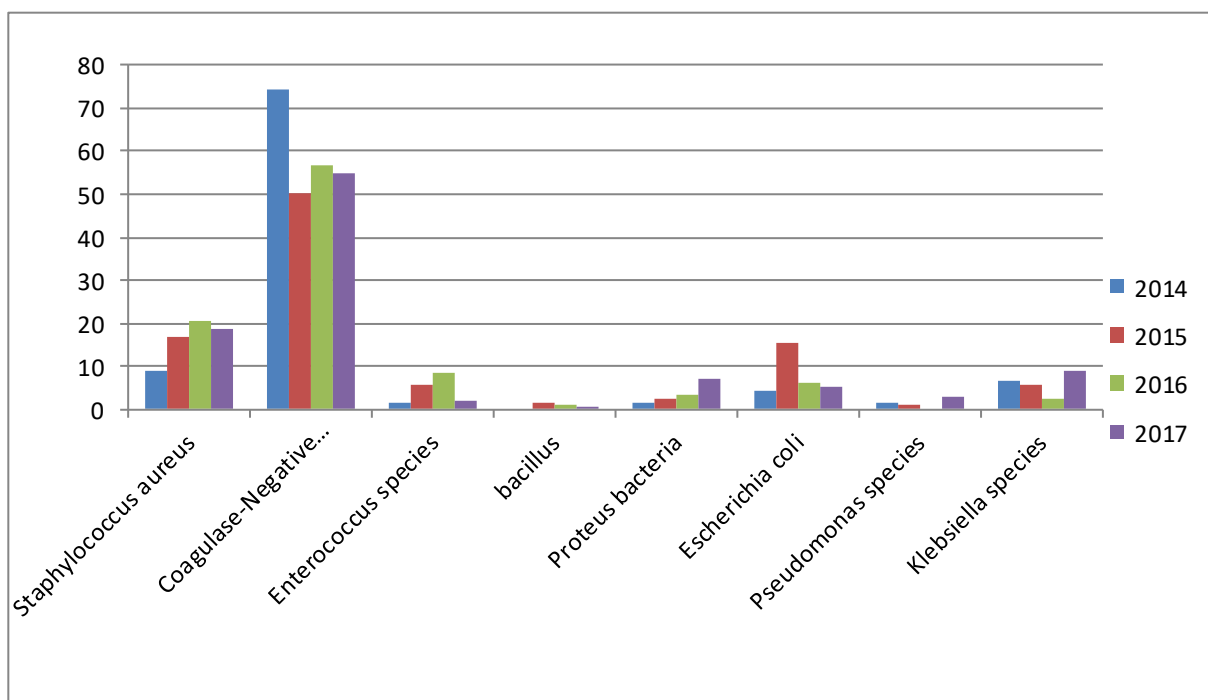


Figure 3: The Bacterial Pathogens Causing Neonatal Sepsis in 2014, 2015, 2016 And 2017

Staphylococcus aureus was with high susceptibility to clindamycin, vancomycin, and Gentamicin (78.3%, 67.4% and

47.8%).

Coagulase-Negative Staphylococci is more susceptible for vancomycin and clindamycin (84% and

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75.1%); *Enterococcus* species shows a high susceptibility to clindamycin (82.6%). While the gram negative bacteria

Escherichia coli and *Klebsiella species*; both are highly susceptible to Imipenem (90.5% and 93.9%) respectively (Table 3).

Table 3: Antimicrobial Susceptibility Results for The Main Bacterial Isolates in All Neonatal Septic Cases

Antibiotic	Micro-Organism				
	<i>Staphylococcus aureus</i> [n=92](%)	<i>Coagulase-Negative Staphylococci</i> [n=325](%)	<i>Enterococcus species</i> [n=23](%)	<i>Escherichia coli</i> [n=42](%)	<i>Klebsiella species</i> [n=33] (%)
Penicillin	1.1	4.6	4.3	4.8	0
Amoxicillin /clavulanic acid	0	9.8	17.4	9.5	9.1
Ampicillin	0	1.5	8.7	0	9.1
Oxacillin	17.4	18.5	4.3	0	0
Piperacillin	0	0	8.7	0	0
Cefixime	0	0.3	13	9.5	18.2
Cephalothin	19.6	42.2	21.7	7	18.2
Cefotaxime	1.1	0	0	4.8	26.1
Erythromycin	5.4	5.8	4.3	0	0
Azithromycin	2.2	3.4	8.7	0	0
Amikacin	10.9	10.5	0	64.3	48.5
Gentamicin	47.8	60.6	34.7	47.6	30.3
Nalidixic acid	1.1	0.9	0	11.9	9.1
Norfluxacin	1.1	0.9	0	12.4	0
Imipenem	6.5	2	13	90.5	93.9
Vancomycin	67.4	84	70	14.3	6.1
Clindamycin	78.3	75.1	82.6	2.4	26.1
Trimethoprim/sulfamethoxazole	1.1	2	0	14.3	18.1

DISCUSSION

The causative pathogens in neonatal sepsis differ from place to place and the frequency of the causative organisms varies from hospitals to others at different times; which make certain that there is a need for continuous looking out to the responsible organisms and their drug susceptibility pattern at a local level.

Gram-positive bacteria were the main causative pathogens, with CoNS alone accountable for more than half of neonatal sepsis cases. This situation differs from that in the developing countries, where CoNS have been shown to be at lower

percentage¹⁵. CoNS have been recorded consistently as the main causative pathogens of LOS in the developed countries and also in Kuwait and other Asian countries¹⁶. Recently there is increasing incidence in care of premature newborns, so those premature infants are extremely vulnerable to less virulent pathogens such as CoNS, which might have contributed to the pattern of LOS seen in the Arab states in the Gulf region¹⁷. Although CoNS have been detected as the most common causative organisms of LOS, they form part of the skin flora and are a common contaminant; distinguishing contamination

from true infection is very challenging. Increasing tendency of clinicians to interpret CoNS blood culture as an infection rather than a contamination; because of the increasing in the population of premature infants in recent years; can be a cause of the current increase in CoNS infections. A study performed in 5 neonatal intensive care units (NICUs) in the following hospitals: Dubai Hospital and Tawam Hospital in the United Arab Emirates, Al Sabah Maternity Hospital in Kuwait, and King Abdulaziz Hospital and the Maternity and Children's Hospital in Saudi Arabia determine nearly a similar finding¹⁸.

The Second most common organism in this study was *Staphylococcus aureus* (16.6%). While another study conducted in Nigeria and in Nepal determines that the *Staphylococcus aureus* the main causative pathogen of the neonatal sepsis^{2,4}. This variation might be due to differences in the study methodology, environmental and host factors that exist.

Regarding gender, at the present study, males were found to be with a higher prevalence of sepsis compared to the female. Other researchers in Nigeria¹⁹, Ethiopia²⁰, Iraq²¹, and Indonesia²² reported a similar finding. The higher prevalence of sepsis in males may be explained by the increased biological vulnerability of males to infection²³. Male sex hormones, androgens, have been shown to be suppressive on cell-mediated immune responses. In contrast, female sex hormones exhibit protective effects which may contribute to the natural advantages of being a female under septic conditions. Thus, the hormonal status has to be

considered when treating septic patients²⁴. However, Omoregie *et al.* found that there is no significant sex difference in the prevalence of bacterial sepsis among young children in Benin City²⁵.

This study revealed a high percentage of the LOS (69.2%). Other study enrolled in Iraq (20) revealed that the EOS was more than that of LOS; it was determined that EOS caused by gram-negative bacteria was more frequent than that caused by the gram-positive bacteria in both EOS and LOS. As well as Studies conducted by Mathur *et al*²⁶ in India and Mokoulo *et al*²⁷. in Nigeria reported a higher percentage of EOS. The factors that have been implicated in the highest incidence of LOS in this study are the poor hygiene, poor cord care and bottle feeding. Therefore, there is an urgent need to enlighten the general public on the proper way of care for the newborn infants.

Regarding the antimicrobial susceptibility results, this study revealed that the gram positive bacteria (Coagulase-Negative Staphylococci and *Staphylococcus aureus*) were with a high susceptibility to clindamycin and vancomycin. Whereas the gram negative bacteria (*Escherichia coli* and *Klebsiella* species; both are highly susceptible to Imipenem (90.5% and 93.9% respectively), similar results were observed by other researcher²⁸ who found that imipenem had a very high effect on gram negative strains. However, it is recommended that imipenem should be used as a last line antibiotic to avoid the occurrence of the microbial resistance. Thus the maximum resistance was seen against Ampicillin, Penicillin, Cefotaxime and Amoxicillin/ Clavulanic acid. On other

hand a study enrolled in west Africa²⁹ found that ampicillin has a moderate rate of antimicrobial resistance. The drug resistance of the microorganisms makes the neonatal sepsis cases to be a rapidly emerging, potential disastrous problem.

This situation is worse in the developing countries because of the lack in the legislation that face the uncontrolled selling of different antimicrobial drugs, shortage of surveillance of the standards of maternity homes and the practices of traditional birth attendants who deliver almost 80% of all births³⁰.

As a result of combination of microbial characteristics and the selective pressure of antimicrobial use; the problem of antibiotic resistant infections in the developing world has increased consistently in the last few decades³¹⁻³⁴.

Accordingly, in 2017 a research conducted in different areas of Nigeria to determine the antimicrobial resistance of Coagulase-negative staphylococci isolated from Nigerian traditional fermented foods, the study revealed that nearly all of the isolates were resistant (93.8%) to Ampicillin, about (84.5%) of the isolates were resistant to trimethoprim-sulfamethoxazole, 42.9% of the isolates were resistant to oxacillin, 60.7% resistant to Augmentin, 6.8% resistant to Cefotaxime, 40.6% resistant to Ciprofloxacin, (28.1) resistant to Erythromycin, and (21,9%) were resistant to Gentamicin³⁵; virtually, when these results compared with that obtained from our study in Hevi Hospital in Dohuk city, it seems to be nearly a close results.

As a conclusion, the most common pathogen causing neonatal sepsis during all four years was Coagulase-Negative

Staphylococci, consisting (58.5%) of cases; followed by *Staphylococcus aureus* (16.6%) and *Escherichia coli* (7.6%). The neonatal sepsis is affecting male more than females (1.5:1). Most of the isolates were resistant to the commonly prescribed Antimicrobial drugs, and many of these isolates showed a resistance for more than 2 of the antimicrobial drugs.

REFERENCES

1. Lawn JE, Cousens S and Zupan J. Four million neonatal deaths: When? Where? Why? *Lancet*. 2005; 365 (9462): 365,891– 900.
2. Peterside O, Pondei K and Akinbami, FO. Bacteriological Profile and Antibiotic Susceptibility Pattern of Neonatal Sepsis at a Teaching Hospital in Bayelsa State, Nigeria. *Trop. Med. Health*. 2015; 43:183–190.
3. Kaistha N, Mehta M, Singla N, Garg R and Chander J. Neonatal septicemia isolates and resistance patterns in a tertiary care hospital of North India. *J. Infect. Dev. Ctries*. 2010;4:055–057.
4. Shaw CK, Shaw P and Thapalia A. Neonatal sepsis bacterial isolates and antibiotic susceptibility patterns at a NICU in a tertiary care hospital in western Nepal: a retrospective analysis. *Kathmandu Univ. Med. J*. 2007; 5:153–60.
5. Pradeep V, Pramod KB, Niranjana N, Sarika S, Prathusha J and Satya N. Neonatal sepsis: epidemiology, clinical spectrum, recent antimicrobial agents and their antibiotic susceptibility pattern. *Int. J. Contemp. Pediatr*. 2015; 2:176–180.
6. Tilahun T, Yibeltal T, Birhanie M, Mequanint F, Tsigiereda D, Amare

- B,et al.Clinical outcome and risk factors of neonatal sepsis among neonates in FelegeHiwot referral Hospital, Bahir Dar, Amhara Regional State, North West Ethiopia 2016. Aretrospective chart review.BMC Res. Notes. 2017; 10:1–7.
7. Shah BA and Padbury JF. Neonatal sepsis: an old problem with new insights. *Virulence*.2014;5,170 – 178.
 8. Du Pont-Thibodeau G, Joyal, JS & Lacroix J. Management of neonatal sepsis in term newborns. *F1000 Prime Rep*. 2014; 6: 67.
 9. Tsai MH, Hsu JF, Chu SM, Lien R, Huang HR, Chiang MC, et al. Incidence, Clinical Characteristics and Risk Factors for Adverse Outcome in Neonates With Late-onset Sepsis. *Pediatr. Infect. Dis.J*.2014;33(1):e7–e13.
 10. Basavaraj P, Jyothi P and Basavaraj M. Bacteriological profile of neonatal septicemia and antibiotic susceptibility pattern of the isolates. *J. Nat. Sci. Biol. Med*. 2013; 4:306.
 11. Downie L, Armiento R, Subhi R, Kelly J, Clifford V, Duke T, et al. Community-acquired neonatal and infant sepsis in developing countries: Efficacy of WHO's currently recommended antibiotics - Systematic review and meta-analysis. *Arch. Dis. Child*. 2013; 98:146–154.
 12. Laxminarayan R, Matsoso P, Pant S, Brower C, Røttingen JA, Klugman K, et al. Access to effective antimicrobials: A worldwide challenge. *Lancet*. 2016; 387:168–175.
 13. World Health Organization. World health report 2005. Make every mother and child count. World Health (WHO Press, 2005). 111-119. Available from: http://www.geopsy.com/memoires_theses/whr2005_en.pdf
 14. Leigue L, Montiani F and Moore BA. Antimicrobial susceptibility and minimal inhibitory concentration of *Pseudomonas aeruginosa* isolated from septic ocular surface disease in different animal species. *Open Veterinary J*.2016; 6(3): 215-222.
 15. Mashaly GE, El-Sabbagh, AM, El-Kazzaz, SS and Nour I. MBL2 & Gene Polymorphism and the Association with Neonatal Sepsis in Egyptian Neonates, a Case Control Study. *Open J. Immunol*.2016;6: 111–119.
 16. Vergnano S, Menson E, Kennea N, Embleton N, Russell AB, Watts T,et al. Neonatal infections in England: the Neon IN surveillance network. *Arch. Dis. Child. Fetal Neonatal Ed*.2011; 96: F9–F14.
 17. Emma SL, Elisabet G and Sara MS. Neonatal Sepsis by Bacteria: A Big Problem for Children. *Clin. Microbiol*.2012; 2(6): 125.
 18. Majeda SH, Abdullah AT, Sameer YA,Hussain B, Anwar K, Laila MA, et al. Late-onset neonatal sepsis in Arab states in the Gulf region: two-year prospective study. *Int. J. Infect. Dis*. 2017; 55: 125–130.
 19. Mahmood A, Karamat KA and Butt T. Neonatal sepsis: high antibiotic resistance of the bacterial pathogens in a neonatal intensive care unit in

- Karachi. *J. Pakistan Med. Assoc.* 2002; 52: 348–350.
20. Amare G, Wubishet L, Feleke M, Beyene M, Belay A, Gizachew Y et al. Bacterial profile and drug susceptibility pattern of neonatal sepsis in Gondar University Hospital, Gondar northwest Ethiopia. *Der Pharmacia Lettre.* 2012; 4(6): 1811-1816.
 21. Jumah DS, Hassan MK. Predictors of mortality outcome in neonatal sepsis. *Med J Basrah University.* 2007; 25(1): 11–18.
 22. Kardana IM. Incidence and factors associated with mortality of neonatal sepsis. *PaediatrIndones.* 2011; 51(3): 144-148.
 23. Petit PL & van JK. Analysis of hospital records in four African countries, 1975-1990, with emphasis on infectious diseases. *J. Trop. Med. Hyg.* 1995; 98: 217–27.
 24. Angele MK, Pratschke S, Hubbard WJ and Chaudry IH. Gender differences in sepsis: Cardiovascular and immunological aspects. *Virulence.* 2014; 5:12–19.
 25. Omoregie R, Egbe CA, Ogefere HO, Igbarumah I and Omijie RE. Effects of gender and seasonal variation on the prevalence of bacterial septicemia among young children in Benin City, Nigeria. *Libyan J. Med.* 2009;4:107–109.
 26. Mathur M, Shah H, Dixit K, Khambadkone S and Chakrapani A. Bacteriological profile of neonatal septicemia cases Irani S. *J Postgrad Med.* 1994; 40:18–20.
 27. Mokuolu AO, Jiya N, Adesiyun OO. Neonatal septicaemia in Ilorin: bacterial pathogens and antibiotic sensitivity pattern. *Afr J Med Med Sci.* 2002; 31(2): 127–130.
 28. Yusef D, Shalakhti T, Awad S, Algharaibeh H and Khasawneh W. Clinical characteristics and epidemiology of sepsis in the neonatal intensive care unit in the era of multi-drug resistant organisms: A retrospective review. *Pediatr. Neonatol.* 2018; 59:35–41.
 29. Bernabé K J, Langendorf C, Ford N, Ronat JB and Murphy RA. Antimicrobial resistance in West Africa: a systematic review and meta-analysis. *Int. J. Antimicrob. Agents.* 2017; 50:629–639.
 30. Mukhopadhyay S and Puopolo K M. Risk assessment in neonatal early onset sepsis. *SeminPerinatol.* 2012; 36: 408–15.
 31. Fair RJ and Tor Y. Antibiotics and bacterial resistance in the 21st century. *Perspect.Medicin.Chem.* 2014; 6:25–64.
 32. AssafiMS, Mohammed RQ and Hussein NR. Nasal carriage rates of *Staphylococcus aureus* and CA-Methicillin resistant *Staphylococcus aureus* among university students. *Int. J. Microbiol. Res.* 2015;5:123-127.
 33. Hussein NR, Assafi MS, and Ijaz T. Methicillin-resistant *Staphylococcus aureus* nasal colonisation amongst healthcare workers in Kurdistan Region, Iraq. *J Glob Antimicrob Resist.* 2017; 9: 78-81.
 34. Habeeb A, Hussein NR, Assafi MS and Al-Dabbagh SA. Methicillin resistant *Staphylococcus aureus* nasal colonization among secondary school

- students at Duhok City-Iraq. J Microbiol Infect Dis. 2014;4:59-63.
35. Fowoyo PT and Ogunbanwo ST. Antimicrobial resistance in coagulase-negative staphylococci from Nigerian traditional fermented foods. Ann. Clin. MicrobiolAntimicrob. 2017; 16(1): 4.

ثوخته

شیوازی بقرطریکرنا ئنتیبیوتیکان و بکتیریاییں دوورخستی د ثیسبونا خوینی یا زاروکی ساظا ل نه خوشخانا هیطی یا زاروکان ل باژیړی دهوکی

شنشین و نارمانج: ثیسبونا خوینی یا زاروکی ساظا ئیکه د بقره لاظترین نقطه قرین مرنا زاروکی ساظا ل ناظا وهلا تیت تیشدکظن. وهقرده ناریشه ل دور طوهارتنا شیوازی بکتیریا مروطی و شیوازی بقرطریکرني. نارمانج د طی طهکولینی بو دیارکرنا ئو بکتیریاییں دبنه نقطه قری ثیسبونا خوینا بضیکي ساظا، بقرطریکرنا زینده وقران بو بکارئینانا ئنتیبیوتیکین بقره لاظ ل نه خوشخانا هیطی یا زاروکان بو بقرور دکرني د ماوی ضوار سالان دا.

ریکین کاری: طهکولینکا ناشوخت هاته ئنجامدان د ماوی ضوار سالان دا. ذوان زانیاریی ب دست کتفین ل نه خوشخانا هیطی یا زاروکان ل باژیړی دهوکی د ناظبقره سالین ۲۰۱۴ تا کو ۲۰۱۷ کو تیدا زانیاریی ۱۰۵۸ نمونیت خوینی ئوین هاتینه هنارتن ژبو صاندنی. ذوان ئنجامان ۵۳۶ ب ئقرینی و ۵۲۲ ب نقرینی دقرضون. ذ ئنجامین ئقرینی ۵۵۵ بکتیریاییں دبنه نقطه هاتنه دیارکر.

نه نجام: د طی طهکولینی دا دقرکفت کو ریذا ره طقری می بو ره طقری نیر ۱:۵:۱ و بقره لاظترین بکتیریا هاتیة جودا کر Coagulase Negative Staphylococcus ب ریذا ۵۸,۵% و ل دیظدا Staphylococcus aureus ۱۶,۶%, Escherichia coli ۷,۶%, ب شیوه کی طشتی ههستیاری ل بقرزترین ناست دا هاته تییینیکر ب ریذا ۳۰% ل سالا ۲۰۱۷ بوو. ض نامار نینن کو ب طرنطی جوداهیی بکفت دناظبقره ذبلی جوری Enterococcus کو بقرزترین ریذه هه بوو بوئیسبونا خوینی یا دواکفتی.

دستکفییین طهکولینی: ئطی قهکولینی دیارکر کو بقرطرتن ددی طهکک ئنتی بایوتیکین بقره لاف دهیة بکارئینان یا زیده بوی کو دبیتة نقطه قری سنورکرنا ههلبذارتین ضارده سقرییت ظان جوړه هه ودانا. ئطه ضنده دی ذ ئنجامی بکارئینانا ئنتی بایوتیکا ب شیوه کی نکهونترولگری و نه بونا ثلان و ریپقرین بکارئینانا ئنتی بایوتیکان د بواری ساخلمیا طشتی دا.

الخلاصة

العزلات البكتيرية وأنماط مقاومتها للمضادات الجرثومية في الانتان الوليدي المسجل في مستشفى هيقي التعليمي بمدينة دهوك/ منطقة كوردستان العراق

خلفية واهداف البحث: الانتان الوليدي يعتبر من اهم الاسباب في موت الأطفال حديثي الولادة في العالم النامي. وهناك تغير مستمر في الأنماط الجرثومية وحساسيتها للمضادات الجرثومية. تهدف هذه الدراسة الى تحديد المسببات البكتيرية للانتان الوليدي، وحساسية هذه الجراثيم للمضادات الجرثومية الشائعة الاستخدام في مستشفى هيقي ومقارنتها بالعوامل المسببة خلال السنوات الاربع الاخيرة.

طرق العمل: أجريت دراسة مرجعية للبيانات المسجلة في أرشيف مستشفى هيقي ولفترة أربع سنوات من عام 2014 حتى عام 2017، تم أخذ ما مجموعه 1058 من الزرع الدموي. وقد كانت عينات الزرع الموجبة والسالبة للجراثيم هي 536 و 522 على التوالي، وعزلت 555 مسبب مرضي من العينات الموجبة للزرع الجرثومي.

النتائج: أوضحت الدراسة الحالية إن نسبة إصابة الإناث إلى الذكور بالانتان الوليدي كانت 1:5:1 وإن أكثر الجراثيم شيوعاً كانت Coagulase Negative Staphylococci (58.5%) و (16.6%) و Staphylococci aureus Escherichia coli (7.6%). تم تسجيل النسبة الاعلى من الانتان الوليدي (30%) عام 2017. لا يوجد فرق احصائي معنوي للجراثيم المسببة للانتان الوليدي المبكر والمتأخر.

الاستنتاجات: أظهرت هذه الدراسة تزايداً في مقاومة الجراثيم للعديد من المضادات الحيوية الشائعة الاستخدام مما يحد من خيارات علاج الانتان الوليدي. وتتجم هذه المقاومة عن الاستخدام غير المسيطر للمضادات الحيوية وفقدان السياسات والمبادئ التوجيهية في الصحة العامة.